Journal of Forensic Medicine
P.O. Box 1010 · Johannesburg · South Africa
Sponsored by the South African Medico-Legal Society
Editor: H. A. Shapiro, Ph.D., M.B., Ch.B., F.R.S.S.Af.

A Comparative Survey of the Control Of Alcoholic Beverages*

The growing public awareness of a social responsibility in connexion with the production, distribution and consumption of alcoholic liquor has led to a plethora of complex legislation in most countries. Moreover, the speed of modern road transport has created new hazards alike for the driver, his passengers and the pedestrian population. This has inevitably led to a vast amount of research into the effects of alcohol on driving capacity and safety. In turn, this has had its effect on legislation which ranges from the arbitrary imposition of penalties based on an alcohol level in the body (irrespective of whether the person concerned was under the influence of alcohol) to a reasonable and just assessment of all the circumstances in a particular case (where the alcohol concentration is regarded as only one datum to be evaluated in the complex of events).

The relation of alcohol consumption to crime, its role as a mitigating factor in certain circumstances and the problem alcoholism as a disease, are other facets of this intricate issue.

It is significant that total or modified prohibition has never had the deterrent effects intended by the propronents of the imposition of austerity and abstinence by decree.

No consideration of the manifold issues raised can be of value unless an authoritative and factual background is available as a basis for discussion. Mr. van Niekerk's *Survey* is therefore of outstanding importance. The author gathered his data at first hand by personally visiting the countries he reports on (the European Countries, including Scandinavia and the United Kingdom, North America, Australasia and the territories of Southern Africa).

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* *A Survey of the Control of Alcoholic Beverages in Other Countries.* By J. A. H. van Niekerk, B.Comm. 1958. (Pp. 254 + Index. 21s.). Johannesburg: Brewers' Institute of South Africa.

His comparative account of the different national legislations highlights the differential treatment accorded the aboriginal populations by the invaders, e.g. 'South Africa remains the only country in Africa south of the Sahara where the native population may not legally purchase European liquor of any kind' (p. 34). Mr. van Niekerk's *Report* also illustrates the futility of repressive measures. Even in homogeneous populations, this policy seems to be disastrous. He finds, e.g. that 'much more alcoholism and drunkenness occur in Norway and Sweden despite their rigid control and relatively low *per capita* consumption than in Italy which has very few restrictions and a high rate of consumption' (p. 24). Again (on p. 104): 'The Swedish experiment in liquor control is an outstanding example of the futility of attempting to alter established habits and customs by means of straight-jacket control, repressive measures without commonsense and humane regard for the desires of the community.'

Some of his statistics are surprising. South Africans, e.g. emerge as 'indisputably the largest consumers of spirits in the world and amongst the smallest consumers of beer' (p. 238).

Because the revision of liquor legislation is almost universal to-day, the author undertook this world-wide survey in order to assist the South African Government in devising effective liquor legislation. His investigation (sponsored by the Brewers' Institute of South Africa) has resulted in a completely objective and unemotional *Report* which provides essential information for a rational approach to the legislative problems facing any country.

Enactments in this field will never be effective unless they are also just and reasonable, taking into account social customs and human propensities of normal persons. The fallacy resides in imposing on the normal population (after all, the vast majority of persons involved) rules more suited to the management of the mentally ill.

It must be appreciated that our law makers are by no means experts in these matters. The qualifications for parliamentarians do not stipulate any special knowledge of social problems. They would therefore be well advised to study the implications of the facts gathered by Mr. van Niekerk before embarking on a legislative programme. This survey will ensure the omission of bias and prejudice in the construction of legislation—if there is an honest desire to cope not with the obsessions of extremists, but with the realities of the situation.

The Prolonged Blood Sugar Curve for Forensic Purposes

Alfred J. Coulthard

From the Laboratories of H.M. Prison Service, London, England

Ever since a defence based upon susceptibility to spontaneous or fasting hypoglycaemia probably saved Derek Lees Smith from execution for the murder of his mother, this possibility has continued to be considered in cases where persons accused of murder claim to have been without nourishment for a lengthy period immediately before the crime, particularly of course if any history of hypoglycaemic symptoms exists.

Since the above case[1] this laboratory has been concerned in an ever-increasing total of investigations into the blood chemistry of persons abstaining from food, from food and drink, or from food with an accompanying intake of alcoholic drinks. The investigation most called for is the Prolonged Blood Sugar Curve, which is basically the normal clinical blood sugar curve extended to 7, 8 or even 12 hours.

Basically, a mere prolongation of the normal clinical procedure is not a satisfactory method, the requirements from the two investigations being really quite different.*

The need for well-spaced but consecutive readings down to a minimum of say 20 mg. per 100 ml. rules out a number of clinical methods. Furthermore, questions of venous or capillary blood, 'true' glucose or total reducing substances, and of photometric or other measurements must all be considered. There are very sound reasons for choosing venous blood, a 'true' glucose method and titrimetric measurement. The grounds for these decisions will be apparent in some cases. In the others it can be assumed that each alternative has had a lengthy trial before the recommended choice was decided upon. The object, of course, is to discover whether in fact the sugar concentration, under the conditions imposed, will fall below say 60 mg. per 100 ml. and, if so, for how long, and to what minimum it does fall (Figs. 1 and 2). Linked to this investigation, other work, e.g. electroencephalography, may well be performed; but these supplementary investigations form no part of this paper; hence the require-

* With clinical methods, too, the recent tendency has been, for obvious reasons, to place more value upon reducing the volume of the blood sample than upon increasing the accuracy or sensitivity of the estimation. The relative values of these two considerations are reversed in the type of investigation with which this paper is concerned.

ment of well-spaced consecutive readings within the range of say 20 to 80 mg. per 100 ml.

All the requirements reduce the methods available to a manageable few and it has been found that the best of these is a modification of King's original 'micro'-method,[2, 3] which was, in turn, based upon the extremely accurate Harding titrimetric method.[4] This is performed with the solutions as originally given (see *Appendix*) and the technique is modified as now described.

i. 2 ml. of the preliminary solution are not sufficient for our purpose and the quantities for producing the protein-free supernatant fluid are amended to:

0.4 ml. of blood;

7.0 ml. of isotonic sodium sulphate/copper sulphate solution;

0.6 ml. of 10% sodium tungstate solution.

ii. A greater spacing or increased sensitivity of readings is required over that provided by the original method, good though that is. This may be achieved in more ways than one but, provided there are no objections, the simplest of all is to increase the final proportions to:

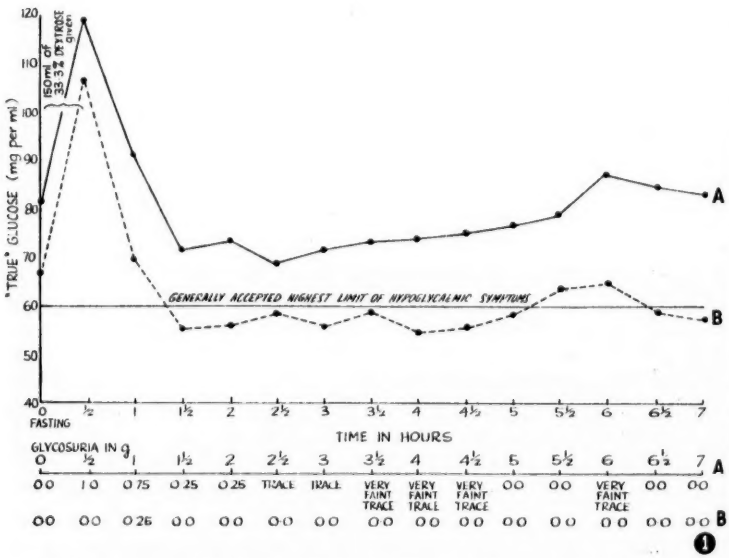

Fig. 1. Prolonged blood sugar curves of prisoners charged with murder. (Venous blood—'true' glucose values, modified King's micro-method).

A: Prolonged blood sugar curve. A normal curve. The Defence plea of hypoglycaemia was not accepted.

B: Prolonged blood sugar curve. This curve was accepted as evidence of hypoglycaemia, and the prisoner, as a direct result, was discharged. The accused's age was 22 years.

4.0 ml. of supernatant fluid;
2.0 ml. of alkaline copper reagent.

Then, by using a 20 ml. automatic burette, graduated in 0.1 ml., each 0.1 ml. will represent a difference of 2.9 mg. per 100 ml. (in accordance with Table 1) or, of course, greater sensitivity still can be obtained by noting the preliminary readings and then employing, after the necessary adjustment, a micro-burette with further sub-division.

Table I: Table of Equivalents with the Modified Micro-Method

Reading of Blank minus Reading of Test	*True Glucose (mg. per 100 ml.)*
00·70	20·30
00·80	23·20
00·90	26·10
01·00	29·00
01·10	31·90
01·20	34·80
01·30	37·70
01·40	40·60
01·50	43·50
01·60	46·40
01·70	49·30
01·80	52·20
01·90	55·10
02·00	58·00
02·10	60·90
02·20	63·80
02·30	66·70
02·40	69·60
02·50	72·50
02·60	75·40
02·70	78·30
02·80	81·20
02·90	84·10
03·00	87·00
03·10	89·90
03·20	92·80
03·30	95·70
03·40	98·60
03·50	101·50

Blanks should be read quite frequently, certainly with each batch of potassium iodide solution and each dilution of sodium thiosulphate. For forensic purposes it is also advisable to include pre-checked glucose standards of, e.g. 25, 50, 75 and 100 mg. per 100 ml. and to read these in parallel with the blood samples in the course of the investigation.

To use these standards a 1% solution of glucose in saturated benzoic acid solution should be carefully checked. This is the Stock Solution. For use dilute 1 ml. to 40 ml. with the isotonic sodium sulphate/copper sulphate solution. This is the Standard Solution.

Of this add 0.1 ml., 0.2 ml., 0.3 ml. and 0.4 ml. respectively to each of 4 tubes containing 2 ml. of distilled water and 2 ml. of the copper

reagent. This will give standards of 25 mg. per 100 ml.; 50 mg. per 100 ml.; 75 mg. per 100 ml. and 100 mg. per 100 ml. respectively.

The calculation is as follows:

As 100 mg. per 100 ml. are contained in the equivalent of 0.1 ml. of blood, the calculation is

Blank − Test × 58 = mg. glucose per 100 ml.

i.e. double the factor for the Test Solutions (where the equivalent of 0.2 ml. of blood is used).

This dilution is selected because pipettes of 0.1, 0.2, 0.3 and 0.4 ml. are more often available than a series of double this capacity. If desired, however, the dilution of the Stock Solution can be made 1 : 80 and double the amounts placed in each tube when the factor (29) will be the same as that for the Test Solutions.

Notes

Although only 0.4 ml. of blood are involved in each individual determination, it is essential for forensic purposes that at least 3 or 4 times this amount be obtained in each case. There are several good reasons for this:

i. Duplicate titrations may be considered advisable or possibly after the preliminary titration as above, a secondary estimation using a microburette might be necessary.

ii. The possibility of the loss of a sample, e.g. by a tube breaking in the centrifuge or the waterbath, is a formidable danger here where repetition of the test may be impossible.

iii. Even if unlikely to be called for, it seems wise to have identical material available. (The prepared sample will remain stable for 72 hours at least and should be stored in the refrigerator.)

The usual security procedures must, of course, be applied.

It is particularly important to be careful that at each 30-minute collection of urine the bladder be entirely emptied. One may be so accustomed to take this for granted that, if it is overlooked, this explanation of the unexpected findings may not be suspected.

Thanks are due to the Commissioners, H.M. Prison Commission, for permission to submit this paper for publication.

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Appendix

Method

Reagents

Stock Glucose Solution: 1% glucose in saturated benzoic acid (0.3%).
Standard Glucose Solution: The above diluted 1 : 40 with isotonic solution.
Isotonic Sodium Sulphate/Copper Sulphate Solution: A mixture of 30 ml. 7% copper sulphate solution and 320 ml. 3% sodium sulphate solution.
10% Sodium Tungstate Solution.
Alkaline Copper Reagent: Mix freshly, equal parts of Harding's 'A' solution (13 g. copper sulphate in 1 litre of water) and Harding's 'B' solution (24 g. sodium potassium tartrate, 40 g. sodium carbonate (anhydrous), 50 g. sodium bicarbonate, 36.8 g. potassium oxalate and 1.4 g. potassium iodate in 1 litre of water).
2% Potassium Iodide: Freshly made.
2N Sulphuric Acid.
N/400 Sodium Thiosulphate: Freshly diluted from N/10.
1% Starch Solution: Freshly made.

Technique

1. Place 7.0 ml. isotonic sodium/copper sulphate in a centrifuge tube.
2. Add 0.4 ml. blood and 0.6 ml. 10% sodium tungstate.
3. Mix well and centrifuge.
4. Add 2.0 ml. of the alkaline copper reagent to 4.0 ml. of the supernatant fluid in a wide blood sugar tube.
5. Add 2.0 ml. of the alkaline copper reagent to 4.0 ml. of the isotonic sodium sulphate/copper sulphate solution for a 'blank'.
6. Add 0.1, 0.2, 0.3 and 0.4 ml. respectively of the Standard Glucose Solution to one each of 4 other tubes containing 2.0 ml. alkaline copper reagent and 2.0 ml. distilled water. These are the Standards.
7. Cover each tube with a large glass marble.
8. Place in a boiling water-bath for 10 minutes.
9. Cool immediately in trough of cold running water.
10. Add 1.0 ml. 2% iodide and 1.0 ml. 2N sulphuric acid. Stand for 1 minute.
11. Titrate with N/400 sodium thiosulphate with one drop of starch as indicator.

Calculations

For the Test:
Reading of Blank − Reading of Test × 29 = mg. glucose per 100 ml.
For the Standards (if dilution is 1 : 40 as above):
Reading of Blank − Reading of Standard × 58 = mg. glucose per 100 ml.

A Case of Criminal Poisoning by Parathion

W. Van Hecke, M.D., A. Derveaux‡ and M. J. Hans-Berteau**

*The Laboratory of Legal Medicine of the University of Ghent**
and
The Laboratory of Pharmacology of the University of Brussels†

Parathion§ has become a frequent cause of acute poisoning, usually lethal. It is already responsible for a considerable number of deaths, mostly in continental Europe. English-speaking countries seem, on the contrary, to have been spared up to now. Most cases are accidental or suicidal. We reported recently[8, 9] 15 cases which had occurred in Flanders, expressing the fear that it could not be long before crimes, too, would be committed in Belgium by means of this compound. At the time, criminal cases had just been reported in Germany and in Austria. In the latter country, Schwarzacher, as far back as 1952, had already investigated more than 30 cases, of which 2, unpublished to our knowledge, were criminal. We had not long to wait before our fears materialized.

It is the object of this paper to relate a murder committed by the administration of Parathion in beer. The interest in the case is enhanced by the fact that the criminal made a complete confession and the details of the crime could therefore be cleared up completely. This case also illustrates the many difficulties which face medical and chemical experts in the general investigation of criminal poisoning.

Case History

At 8.50 p.m. on 21 August 1956, the police were informed that A. W., a youth aged 18 years (who had previously enjoyed excellent health) had died suddenly at a fair. Witnesses were immediately questioned, among them F. W., the brother of the deceased. The latter said that he had seen his brother at 8 p.m., when he was sitting on the side of the road very ill

* Director: Prof. F. Thomas.
† Director: Prof. L. La Barre.
‡ Docteur en Pharmacie.
** Pharmacien, Licenciée en Chemie.

§ Parathion (00-diethyl-0-o-nitrophenyl-thiophosphate was synthesized by the German chemist Schrader, in 1944. It is an extremely potent insecticide which is marketed by Bayer under the name E605. E605 *Forte* is a 50% concentration and is sold in bottles, the contents of which have to be diluted before use in agriculture.

Absorption can occur by all routes. Pharmacologically its action resembles that of physostigmine, but is of much longer duration. It inactivates acetylcholinesterase. The antidote is atropine sulphate.

and complaining that someone had put petrol in his beer, that he could not see any more and that he felt as if he was losing his senses. A doctor was called at once but, when he arrived at 8.30 p.m., the youth was dead.

The innkeeper in whose establishment the beer had been consumed, made the following statement:

A. W., in the company of another youth whom he did not know, had ordered beer for them both. Hardly had A. W. swallowed a draught (about half his glass) when he complained that the beer tasted of petrol.

(It had originally been stated during the inquest that the innkeeper thereupon himself sipped from A. W.'s glass without noticing anything abnormal. This version turned out to be wrong. The beer the innkeeper tasted was beer he had poured from the tap in a fresh glass).

In the meantime A. W. ran outside, where he vomited, the poisoner availing himself of the opportunity to put the victim's glass surreptitiously in his pocket, walk out of the inn and get rid of it.

Other witnesses were able to give further information. The victim had followed a local bicycle race during the afternoon. He was seated in a car, of which they were able to give a description, driven by a young man who was unknown to them. This young man had remained in A. W.'s company until he became sick. A partial post-mortem examination was carried out locally the next day. The findings (Dr. J. Piret) can be summarized as follows:

The body was that of a rather lean man whose external examination revealed nothing abnormal and no traces of trauma. Slight mydriasis.

Brain: No meningitis. No haemorrhage. A peculiar smell reminiscent of 'bad' lavender water, not of petrol.

Chest: Subpleural haemorrhages on anterior surface of the right lung. A certain amount of lung congestion. Bloody froth and mucus in the trachea.

Heart: Although contracted, it had a normal appearance.

Abdomen: The stomach was large and dilated with zones of local congestion. It contained 1 litre of food in the course of digestion, giving off an aromatic smell other than that of gasoline.

Kidneys, Liver: Negative.

Stomach contents and blood were collected for chemical analysis and the stomach wall for histopathological examination, which revealed no microscopic changes whatever apart from marked congestion of the sub-mucosa.

Conclusion: The death must be attributed to acute poisoning by a toxic substance giving the stomach contents and brain a peculiar smell.

Toxicological Report: 1

Only stomach contents, vomited matter and blood were available.

The classical procedure for the detection of poisons was carried out, viz. successive distillation in acid and alkaline media, detection of volatile poisons, extraction in acid and then alkaline medium of the remaining distillation fluid, qualitative deter-

mination of alkaloids, glycosides and metals, after destruction in sulphuric–nitric acid and in chlorate medium. Furthermore, special detection procedures were carried out on the distillate, its residue and the ignited residue.

Sodium hydroxide was added to the alkaline distillation fraction of the stomach contents and vomit. This resulted in a yellow colouring of the 2 samples, revealing the presence of p.nitro-phenol. The blood fraction was negative.

From the foregoing it was assumed that the poison was Parathion. No barbiturates, alkaloids or metals could be detected.

Quantitative measurements were carried out using the calorimetric technique. After ignition of the residue and redissolving it in nitric and then sulphuric acids, a standard procedure was used in the determination of phosphorus, a wavelength of 6500Å being applied. By this method 3.8 g. of Parathion was estimated to be present in the stomach contents and vomited matter.

Conclusion: The poison is Parathion. The amount present in the vomitus and the stomach contents together is 3.8 g. This amount is sufficient to cause rapid death.

The blood alcohol estimated by the Widmark method was 1.2 g. per mille.

The police quickly succeeded in identifying and arresting the unknown youth thanks to the description of his car.

J. B. was 23 years old, and a florist by profession. On 20 September 1956 he made the following statement:

He had met the victim, who was until then unknown to him, at the fair. A. W. had suggested that they should follow the race in J. B.'s car, in compensation for which A. W. would pay him 100 francs. When the race was over at 6 p.m. they went together to have a glass of beer. A. W. had complained to the innkeeper that either the glass must be unclean or that there must be something in the beer. They left each other at 6.15 p.m. and he had driven away. Later he came back and had met A. W. in the street at 9.30 p.m. looking quite well.

On 24 September, he made a new statement declaring that he would now tell the truth. He had, he said, emptied half a bottle of Lirothion (which is a 15% dilution of Parathion sold under a different trade name) which he carried with him and he knew to be dangerous, into the beer of the victim. He never thought A. W. would drink it because of the smell, but he did. He did not attempt to stop him and left hurriedly before the appearances of the first symptoms of poisoning.

The same evening he learnt of the death of A. W., whereupon he had thrown the bottle into the sewage.

Fortunately this bottle was recovered. It still contained 3.5 ml. of Parathion at a concentration of 16% (see below). In his car a larger bottle, labelled 'Lirothion', was discovered. It was from this that he had filled the smaller one.

Toxicological Report: II

The large bottle labelled 'Lirothion' found in the car was next submitted for analysis. The qualitative determinations were carried out as before. The same results were obtained as in the stomach contents and vomited matter. The quantitative determinations were also carried out as previously.

Conclusion: The large bottle contains a 16% concentration of Parathion.

Toxicological Report: III

The small bottle found in the sewage had a capacity of 13 ml. and still contained 3.5 ml. of solution. The quantitative analysis carried out, as mentioned before, yielded 16.3% expressed in Parathion. Using the colorimetric method with sodium hydroxide this figure was 17%.

Conclusion: The small bottle contains Parathion at a concentration of approximately 16%.

Discussion of the Three Toxicological Reports

Assuming that, at the time of the incident, the bottle was completely filled, the conclusion could be drawn that the amount of Parathion ingested was 1.5 g., at the most. This figure differed considerably from that previously advanced. The difference was probably to be explained by the phosphorus contained in the food eaten shortly before death, viz. bread and potatoes, the phosphorus of which is unavoidably estimated as Parathion. One must not forget that the quantitative analysis actually depends on phosphorus and not on Parathion as such.

On reviewing the whole sequence of these chemical investigations critically, it became obvious that the proof that the stomach contents of the deceased contained Parathion at all was not conclusive, in spite of the considerable efforts taken. As has been shown previously in a discussion of the analytical methods for the detection of Parathion intoxication,[8,9] this was to be expected. Further investigations remained indispensable to attain this end, the missing link having to be provided by biological tests destined to show up the anti-acetylcholinesterase activity proper to such compounds.

It was decided to undertake these investigations not only on the residue of the stomach contents and blood which had wisely been set aside, but also on new material to be provided by an exhumation of the body. This was therefore carried out by one of us (Dr. W. Van Hecke) three months after the death of the deceased on 21 November 1956.

Report of the Exhumation

The body was contained in a wooden coffin. Decomposition was already far advanced and the skin covered with mould. The abdominal and thoracic cavities lay open. All the viscera were found in place except the stomach and, as far as one could tell with the amount of putrefaction present, they showed no gross lesions or abnormalities. The neck (which had not been dissected during the first post-mortem examination) was examined but nothing abnormal was found. The skull had partly been opened previously and only the frontal lobes of the brain removed. This dissection was therefore completed, but only putrified matter was found.

The following organs were removed: brain, tongue with pharynx attached, both kidneys, bladder, part of the liver, small intestines, skeletal muscle and heart.

Toxicological Report: IV. Biological Assay

Preparation of the Extracts: From the material obtained at exhumation only extracts of liver, small intestines, and tongue and pharynx were prepared. In addition, extracts of the stomach contents and blood obtained at the first post-mortem examination, were made.

The samples were treated in a mixing blender and macerated with ethyl alcohol in a tartaric acid medium. Twenty-four hours later the solution was filtered and the alcohol distilled under vacuum. Alcohol at 96° was added to the precipitate and the solution filtered off and evaporated. This procedure was repeated 4 times, after which absolute alcohol was used and finally benzine *pro analysis*. The benzine solution was evaporated, 50 ml. of water added and an extraction performed 3 times with 50 ml. of ether. The ether solutions were evaporated to dryness and the extract redissolved in alcohol at 96° and diluted up to 10 ml. in a volumetric flask. This stock solution was used for further biological determinations which were performed by the classical method of Morand and Laborit.[4]

Table 1: Results of Biological Determination of the Anti-Acetylcholinesterase Activity

Extract of	*Acetyl-cholinesterase Activity* (%)	*Loss of Acetyl-cholinesterase Activity* (%)	*Reaction*
Normal plasma	100	0	++
Plasma + liver	88	12	±
Plasma + small intestine	86	14	±
Plasma + stomach contents.. ..	0	100	++
Plasma + tongue and pharynx ..	83	17	±
Plasma + blood	98·5	1·5	—

The experience acquired previously has shown that only the reaction given by the stomach contents is significant. We have explained that values under 20% are too doubtful to be taken into account.

Discussion

This case teaches us little from a clinical point of view, the data being scanty and provided only by non-medical witnesses.

So violent is the poison that the abundant vomiting did not save the victim's life. He died within an hour after ingesting the Parathion.

Parathion is extremely toxic. The lethal dose for an adult, which had at first been estimated at 1.5 g. of E605 *Forte*, is (as a matter of fact) lower,

and of the order of less than 1 g., which corresponds to less than 0.5 g. of the pure compound. A German doctor, who was foolish enough to experiment with it on himself, absorbed a total dose of 230 mg. in the space of a few days. He paid for his daring with his life.

According to our calculations, the total amount of pure Parathion in A. W.'s glass was of the order of 1 g. (± 7 c.c. of Lirothion). He only had one draught, so that he probably did not ingest more than a third to half a gramme of the poison. This quantity, however, proved enough to kill him.

From a pathological point of view, special emphasis must be laid on the very peculiar chemical smell of the blood and organs which is always present unless masked by putrefaction. This smell is due to the solvent and therefore varies. This explains the different ways in which it has been described, viz. as reminiscent of xylol, petrol, turpentine or rancid butter.

Apart from the smell, the findings are mostly negative. The German authors mention a marked softness of the brain which must have escaped our attention. In a case reported by Böhmer[1] (and summarized by us below) a haemorrhage in the pons had led the first investigation astray. Histology is of no avail.

From a chemical point of view we wish to repeat what we have emphasized before, i.e. that chemistry does not identify Parathion as such, and only the complementary biological test is conclusive. This is the reason why, in the present case, part of the work had to be started all over again.

This case confirms what two of us, in collaboration with Heyndrickx and Thomas, were the first to bring out, viz. the possibility of unexpected and long resistance of the poison to hydrolysis. This resistance (at first categorically denied by German chemists) has been confirmed recently by Machata.[3] The biological assay was still 100% positive in the stomach contents of A. W., which had been set aside, 4 months after death.

In the literature, we have found reference to only 5 crimes committed by the administration of Parathion. The first case, to our knowledge, is that reported by Seifert[7] in 1954. This author relates a Machiavellian crime committed on Christmas Eve, 1953. The circumstances were as follows:

A natural child, 9 weeks old, died suddenly. At 9.30 p.m. it had received its last meal. When the father came in unexpectedly at 10 p.m., it slept quietly in the room of the mother. He went alone to the child. Ten minutes later it began to cry loudly, upon which the mother asked him to go and see it again. He came back saying it had vomited. The mother thereupon rushed to the child and found the face pale and the limbs flaccid and drooping. It had obviously vomited repeatedly. The chest and right

side were wet, the bed linen was dirty. The child's face was immediately washed with cold water and the chest rubbed with alcohol.

A doctor, who had in the meantime been called, made an intracardiac injection but without avail. His opinion was that the case was one of natural death from heart failure.

The father gave everyone the impression of great bereavement.

The post-mortem examination did not reveal the cause of death and a toxicological examination was therefore carried out.

In the meantime, the inquest revealed that the father intended to marry another woman and that, as an agriculturist, he was acquainted with the properties of E605. This substance was therefore also looked for. It turned out to be present in the blood, the vomitus, the stomach, the intestinal contents, the liver and the spleen.

Shortly after these findings, the father made a statement to the effect that he had put 2 or 3 drops of E605 on the comforter of the baby and had then pushed it into its mouth. Experiments proved that the quantity of E605 thus fixed on the comforter must have been of the order of 50–100 mg. of E605, corresponding to from 25–50 mg. of the pure substance. Part of this quantity had been eliminated by vomiting.

The still little-known homicidal and suicidal potentialities of the new compound unfortunately received wide publicity when, in 1954, a woman was charged, in Worms, with the murder of a number of members of her family whom she had poisoned by means of chocolates in which she had concealed the compound. To our knowledge, this series of cases has, as yet, not been related.

The second case, chronologically, is that of Pribilla.[5] A man, 48 years old, who for a long time had been on bad terms with his wife, attended an afternoon party at home. All the participants had been drinking freely. His wife gave him a cup of coffee at about 5.30 p.m. A lady who sat next to him noticed that the coffee had a peculiar colour and smell, and the husband made some remark to the same effect. Shortly after drinking it, he was unwell and felt he was going to vomit. He retired to his room where his wife found him dead the next day.

A doctor certified the case as one of natural death by heart failure.

The afore-mentioned lady, who knew about the Worms murder cases through the newspapers, made an experiment. She poured a little E605 in some coffee, whereupon she immediately noticed the same tell-tale discoloration and peculiar smell which had attracted her attention at the party. Thereupon the police received an anonymous letter.

A search in the house of the deceased led to the discovery of 4 little flasks of Parathion which had all been opened. The quantity of Parathion missing was 1.5 ml.

The post-mortem examination was carried out 3 days after death. Parathion was detected chemically in the stomach and intestinal contents, and also in the muscles.

The widow was put under arrest. She energetically denied the charge against her.

The helpful lady was now subjected to an experiment. After her eyes had been bandaged, 6 cups of coffee, to one of which 1.5 ml. of E605 had been added, were placed in front of her. Without hesitation, she pointed out the cup with the poison. The widow was acquitted, however, because the possibility of suicide was considered not to have been completely excluded.

The third case is that of Böhmer[1] already referred to. A woman, aged 32, made a statement to the effect that her husband and herself decided at 4.30 p.m. on 21 April 1954, to obtain a divorce. After this, she said, they both went to a drugstore and bought a bottle of poison. On the way she also bought a quarter of a pound of minced meat. They were back home at 6.30 p.m. While her husband was in the garden, she divided the meat into halves. To the first (intended for her husband) she added the poison, but not to the other (because, she explained, she was afraid to die). They ate together. He had spread his meat on a slice of bread. After eating it he shivered, and at 8.30 p.m. he went to bed complaining of headache. She joined him and took him in her arms. He vomited, and she then became frightened and hurriedly called a doctor who, when he arrived, found her husband already dead. She threw the empty bottle away in a dustbin. Later, she admitted that she had, in fact, bought the poison in the absence of her husband.

The post-mortem examination revealed an extensive haemorrhage in the pons Varolii, with a large amount of blood in the ventricles. The pathologists, therefore, attributed their finding to hypertension and accepted it as being the primary cause of death, to the exclusion of poisoning. However, a toxicological analysis was ordered by the authorities. This revealed the presence of Parathion in the stomach contents, the blood, the urine and the brain.

Lastly, two cases, one of murder and another of attempted murder, have just been brought to our knowledge by Schwachtgen,[6] of Luxemburg. He only mentions them, however, without going into any detail, in a general review of Parathion poisoning meant for the general practitioner.

From a criminological point of view, this affair brings to mind the *perfect crime* dear to the novelist. As one is entitled to fear that the ease with which the compound can be acquired (it is nowadays available on the market in bottles of 15 c.c.) may encourage its criminal use, we have considered it worthwhile to carry out a series of experiments, the results of which are grouped in Table 2.

In the first experiment we simply repeated the gesture of J. B. when he poured the Lirothion into A. W.'s beer. The ensuing mixture, we found, had such a repulsive smell and turbid appearance that one can hardly understand why A. W. only noticed it *after* he had tasted it, and not whilst bringing his glass to his lips. The only reasonable explanation, in our opinion, is that he was under the influence of alcohol, which is confirmed by the blood alcohol value of 1.2 g. per 0/00.

In the second experiment, we replaced the Lirothion by E605 *Forte*. In this instance, although the beer lost its transparency, the smell was much less pronounced. The third experiment was, as a matter of fact, meant to verify an interesting statement by Seifert.[7] This author reports a case where a person, bent on suicide, poured two or three times the lethal dose of E605 *Forte* into 100 c.c. of Schnapps 40%. He drank about half of it, but was only slightly sick. This the author attributes to the fact that the poison was precipitated to the bottom of the bottle. We therefore poured the same poison, in the same quantity, into 100 c.c. of gin of poor quality. The latter became turbid, but there was hardly any perceptible smell and the liquid regained its transparency when pure alcohol was added to it. The biological assay contradicted the explanation advanced by Seifert, as a glance at Table 2 shows that all levels of the liquid proved equally and highly toxic.

Table 2*

Samples Containing the Pseudocholinesterase	*Volume of Sample Used (ml.)*	*Substrate* M/100 *Acetylcholine Solution (ml.)*	*Volume* in ml. of M/100 NaOH *Necessary to Neutralize the Liberated Acetic Acid*	*Acetyl-cholinesterase Activity* (%)		*Loss of Acety-cholinesterase Activity* (%
1. Normal dog plasma	0·5	10	4·64	100		
Plasma + 20% beer	0·5	10	3·72	80·2		
+ 50% beer	0·5	10	3·20	69	100	
+ 50% beer containing 2·36% Lirothion						
(*a*) Sampling at the upper layer of	0·5	10			24·7	
(*b*) Sampling in the middle layer the	0·5	10			16·8	80
(*c*) Sampling at the bottom layer glass	0·5	10			20	
2. Normal dog plasma	0·5	10	4·02	100		
Plasma + 50% beer	0·5	10	2·81	70	100	
+ 50% beer containing 0·71% E605 *Forte*	0·5	10	0·43		15·3	85
3. Normal dog plasma	0·5	10	3·74	100		
Plasma + 20% gin	0·5	10	2·95	78·8	100	
+ 40% gin	0·5	10	2·50	66·8		
+ 20% gin containing 4% E605 *Forte*						
(*a*) Sampling from the upper layer of the gin	0·5	10	0·19		6·4	94
(*b*) Sampling from the bottom of the the glass 1 minute after the addition of E 605 *Forte*	0·5	10	0		0	100

* The assays were likewise carried out by the method of Morand and Laborit. In each experiment, 45 minutes elapsed between the mixing of plasma with beer or gin and the estimation of the acetylcholinesterase activity. They were repeated by the test of Davies and Nicholls[2] on dog whole blood (true and pseudocholinesterase), with identical results.

From these simple experiments it is obvious that the addition of E605 *Forte* in a drink, such as a cocktail or liquor, would have every chance of passing unnoticed, and that the above-mentioned fears are therefore amply justified.

Our case report illustrates the difficulties and set-backs medical and toxicological experts must expect to face when investigating any case of criminal poisoning.

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Non-Traumatic Subdural Haemorrhage

Following Superior Sagittal Sinus Thrombosis

Report of Two Cases

I. Robertson, M.B., Ch. B. (Cape Town)
(Assistant Government Pathologist, Durban)
and
S. S. Grové, F.R.C.S. (Eng.)
(Registrar in Pathology, Natal Provincial Administration, and the Faculty of Medicine, University of Natal, Durban)

Subdural haemorrhage occurring as an unexpected post-mortem finding is a common and important problem. In spite of the absence of a history of head injury or other signs of trauma, police investigations sometimes reveal that head injury did, in fact, occur at a time consistent with the features of the haemorrhage. Depending on the circumstances surrounding the injury, criminal proceedings or compensation claims may be instituted. The forensic importance of such a finding is therefore considerable, and natural causes of subdural haemorrhage must be excluded.

This paper describes two cases of thrombosis of the superior sagittal sinus, in one of which there was subdural haemorrhage. The other case indicates the probable pathogenesis of the haemorrhage and illustrates the common autopsy findings in superior sagittal sinus thrombosis.

Case I

History. An African woman aged 26 years was admitted to hospital unconscious. A relative said that the patient had complained of cough and pain in the chest for 6 months. Fourteen days before admission she developed a headache, poor vision and weakness in the right arm and leg. Three days before admission she suddenly became unconscious while sitting at home. On enquiry no history of head injury could be obtained.

Physical Examination. The patient was unconscious. The pupils did not react to light and were unequal in size. Muscle tone was increased in the limbs, and the tendon reflexes were exaggerated, although equal on both sides. Both fundi were normal. There was marked emaciation and tuberculous cavitation of both lungs was diagnosed.

Laboratory Investigations: A moderate normochromic anaemia was present, with a polymorphonuclear leucocytosis. The white cell count 17,000 per c.mm.; E.S.R. 60 mm. in the first hour (Westergren).

The blood urea was 26 mg. per 100 c.c.
Blood proteins: Total 5.8% (Albumin: 1.1%; Globulin: 4.7%).
Cerebrospinal Fluid: Clear and colourless.
Red cells: 480 per c.mm.
Polymorphs: 2 per c.mm.
Chlorides: 780 mg. per 100 c.c.
Protein: 10 mg. per 100 c.c.
Sugar: 84 mg. per 100 c.c.

Fig. 1. Superior sagittal sinus (Case 1) containing ante-mortem thrombus. The sinus was opened immediately after the calvarium was removed.

Progress. The patient was treated with streptomycin, I.N.H. and penicillin, and tube-feeding was commenced. She did not improve. Her temperature rose and was 104° F. on the sixth day after admission, when she died.

Post-Mortem Findings. Both lungs showed extensive caseous tuberculosis, with cavitation in the left upper lobe and bronchopneumonic infiltration of the right lower lobe.

An unexpected finding was a subdural blood clot $\frac{1}{4}$ inch thick over most of the surface of the right cerebral hemisphere. There was no meningeal staining or exudate and a membrane had not formed. There was no injury to the skull, and dissection of the scalp revealed no wounds or bruises.

An ante-mortem thrombus filled the superior sagittal and both transverse sinuses (Fig. 1). The bridging veins were distended but not thrombosed. No ruptured cortical vein could be seen.

The brain showed no abnormality. In particular, the convolutions were not flattened and no haemorrhages were observed in the substance of the brain.

Histological Examination. The brain showed marked oedema and congestion of cerebral vessels. Capillary thrombi and microscopic capillary haemorrhages were present in both white and grey matter. The ante-mortem thrombus of the superior sagittal sinus contained numerous polymorphs but special stains did not reveal any organisms in it.

The presence of caseous pulmonary tuberculosis was confirmed.

Case 2

History. An adult Indian female was admitted to hospital two weeks after giving birth to a normal infant. The day before admission she had gone to the toilet and, on her return, her husband noticed that she behaved strangely. She laughed, twisted her clothes and jerked her arms and legs. She then had a convulsion followed by coma for a few hours. Consciousness subsequently returned, but it was noticed that she could not speak.

There was no history of head injury.

Physical Examination. The patient appeared conscious, but was unable to talk. She resented flexion of the neck, and all 4 limbs were spastic. The plantar reflexes were extensor. There was a generalized reduction in sensation to pain. There was slight blurring of the right optic disc. The uterus was felt 3 fingers above the symphysis pubis.

Laboratory Investigations. Lumbar puncture showed a cerebro-spinal fluid under a pressure of 180 mm. H_2O. No block was present. Globulin and protein were markedly increased but no cells were reported in the fluid.

Progress. The patient was treated with antibiotics and medical decompression, the tentative clinical diagnosis being superior sagittal sinus throm-

bosis. The following day, however, her temperature was 104° F., and she died 16 hours after admission.

Post-Mortem Findings. Thrombosis of the superior sagittal sinus was present. A striking feature was the enormous distension and thrombosis of the bridging veins (Fig. 2). The cortical veins were also greatly distended. There was subarachnoid haemorrhage, and numerous haemorrhages in the left frontal and right parietal regions of the brain, resembling areas of traumatic cerebral contusion (Fig. 3).

No fracture of the skull was present and careful dissection of the scalp revealed no signs of injury.

The uterus contained septic products of conception.

Histological Examination. There was a large organizing thrombus in the superior sagittal sinus. Organisms were not seen in Gram-stained sections. There was subarachnoid haemorrhage and haemorrhages of varying sizes were seen in the brain substance.

Organizing thrombi were seen in numerous vessels in the uterine wall.

Discussion

Trotter[1] drew attention to the traumatic origin of pachymeningitis haemorrhagica interna. Since then trauma has been correctly emphasized as a cause of chronic subdural haemorrhage. There was a history of head injury

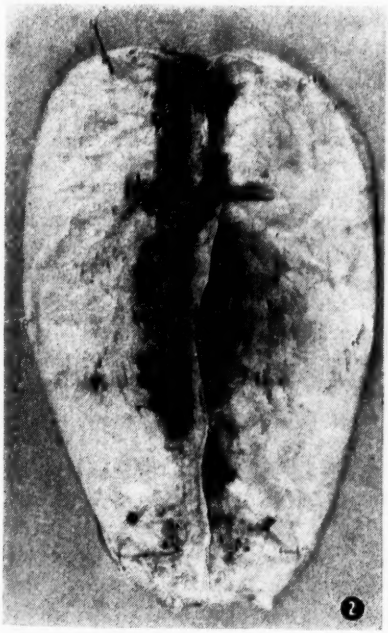

Fig. 2. Undersurface of dura mater (Case 2) showing markedly distended bridging veins containing antemortem thrombus.

Fig. 3. A portion of the temporal lobe of the brain (Case 2), showing an appearance resembling cerebral contusion. Note also the distended cortical veins.

in the vast majority of 50 cases described by Leary.[2] We have seen numerous cases of subdural haemorrhage occurring as the result of birth trauma in infants and following head injury in adults, the latter after a varying latent interval. Martin[3] considered that there would always be a history of head injury in cases occurring in young people; but that, in the elderly, the trauma might be quite trivial and easily forgotten during the lucid interval which might follow it.

The reason for this is indicated by Keegan.[4] He described a case of chronic subdural haematoma in which he observed bleeding from the superior sagittal sinus at operation. He concluded that the anatomical disposition of those cortical veins which bridge the subdural space to enter the superior sagittal sinus, renders them particularly susceptible to rupture by head injury, and that this is the source of most subdural haemorrhages. He considered also that cerebral atrophy would heighten this susceptibility by stretching these veins as the cortex recedes.

This explanation probably accounts for the higher incidence in the elderly and in patients with mental disease and cerebral atrophy, as described by Allen, Moore and Daly.[5]

It seems reasonable to assume, therefore, that head injury is the commonest cause of subdural haemorrhage; that the common source of the haemorrhage is a ruptured bridging vein; and that it may sometimes be caused by trauma so slight that the patient may have forgotten it before symptoms appear.

Slight trauma to the head may cause minimal damage to the scalp, or none at all. Bruises of the scalp are easily missed on clinical examination, and may be seen in fatal cases for the first time at autopsy, when the aponeurotic surface is examined.

Hence the pathologist may be the first to realize that death following sudden cerebral symptoms is due to traumatic subdural haemorrhage, and it may only be on his initiative that police investigations are commenced. It is therefore important that he exclude natural causes of subdural haemorrhage when this condition is found unexpectedly.

Subdural Haemorrhage from Natural Causes. Subdural haemorrhage occurs quite commonly when a large intracerebral haemorrhage of any etiology ruptures through the pia-arachnoid. The arachnoid may also be torn by sudden haemorrhage from an aneurysm of the circle of Willis. Tumours invading the arachnoid or the dura may also cause subdural haemorrhage.

In all these instances the source of the haemorrhage is usually apparent. It seems, however, that other natural causes of subdural haemorrhage exist, in which the underlying lesion is less apparent. Gilman and Tanzer[6] describe a case in which a child 16 months old developed, apparently spontaneously, a subdural haemorrhage whilst in hospital with scurvy. They also found seven other recorded cases in typical scorbutics.

Subdural haemorrhages in blood dyscrasies also occur.

Bailey and Haas[7] and Scott[8] described 2 cases of subdural haemorrhage associated with superior sagittal sinus thrombosis in infants, but this combination of lesions is apparently uncommon in adults.

In the first case described here, we feel justified in assuming that the cause of the subdural haemorrhage was superior sagittal sinus thrombosis, and not head injury. This supposition is supported by the second case, which demonstrates the enormous distension of bridging veins which can result from sagittal sinus thrombosis. The ease with which such distended veins may rupture spontaneously can readily be visualized. Thrombosis of dural sinuses is not uncommon in the severely debilitated and in patients suffering from severe infections. The pathogenesis of the thrombosis in the cases we have described was not clearly established, but the possibility of septic thrombosis cannot be excluded.

These cases emphasize the importance of careful examination of the superior sagittal sinus whenever subdural haemorrhage is found unexpectedly at autopsy.

Summary

Two cases of superior sagittal sinus thrombosis in adults are described. The first case had a spontaneous subdural haemorrhage. The second case suggests the pathogenesis of this haemorrhage.

The forensic significance of subdural haemorrhage due to natural causes is discussed.

We wish to thank Dr. S. Disler, Medical Superintendent, King Edward VIII Hospital, Durban, and the Secretary for Health, South Africa, for permission to submit these cases for publication.

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Duodenal Ulceration in Cases of Burns

Zora Stajduhar-Djuric

Institute of Forensic Medicine, University of Zagreb, Zagreb, Yugoslavia

Intestinal ulceration is a recognized complication of burns of the skin. The site of election of these ulcers is the duodenum, though they occur but rarely. First described by Curling, they are usually known as Curling's ulcers.

There is no one single accepted theory of the causation of death in cases of burning, for many factors clearly participate, but that of toxaemia is widely held. In addition to the obvious trauma involved in a burn, the body is subjected to 3 particular pathological factors, on which the course and length of invalidism depend. These factors are shock, toxaemia and infection. Shock is evident within a few hours of the burning and is due to loss of fluid, toxaemia and pain. During the first few hours of shock irreversible changes may occur in the body, notably in the kidney, the liver or the cardio-vascular system. Consequently shock is a frequent cause of death in the first 2 or 3 days.

Later, for about 5 days, it is the signs of toxaemia which are dominant. Other pathological changes occur concurrently, e.g. those seen in the kidney, the digestive system, as well as biochemical alterations in the serum proteins, carbohydrates, electrolytes and plasma pH, and in vitamin utilization. Toxins, probaly polypeptides, are formed in the body, and there is direct absorption from the burned area of broken-down proteins. These foreign substances enter the blood stream and produce deleterious effects upon a wide variety of vital organs.

On the basis of experimental work, Christopher postulates the presence in the blood stream of specific neuro-toxins. The changes which then appear in the kidneys, in the digestive system and in the blood may be secondary to such central damage.

Hilgenfeld considers that there is an absorption of altered protein from the burned area which contributes to death in those succumbing within a few hours of burning. Animal experiments showed that surgical excision of the affected area, if accomplished within the first 8 hours, prevented the onset of this toxaemia.

On the other hand Marchand, quoted by Pietrusky, does not believe that toxins are demonstrable in those dying rapidly from burns. However, Hilgenfeld and Fasal, supported by an exhaustive survey of the literature, maintain the more popualr theory of intoxication.

More recently changes in blood volume have been incriminated. Underhill, Harrison, Kapsinov and Blalock all believe that there are 2 factors

involved: one, an increase in capillary permeability; the other, a reduction in the osmotic and the other pressures upon which resorption of fluid depends. Not being able to demonstrate any specific toxins, they maintain that in primary shock there is a relative diminution in blood volume, and in secondary shock a true reduction in volume.

Infection is always serious, both in its local and in its general effects, the general condition and resistance of the patient being significantly lowered by such infection. A variety of organisms may be responsible, Staphylococci, *Streptococcus haemolyticus, Ps. pyocyaneus, B. proteus* and coliform bacilli being common examples.

At autopsy on a case of burns there may be no specific changes. Haemorrhages into the mucous membrane of the digestive tract, haemorrhage into or degeneration of the suprarenals, haemorrhage or necrosis of the kidneys, liver or myocardium may all be found. Intestinal ulceration, particularly that of the duodenum, must be rare.

In 1842 Curling described 12 cases of burns of the skin which were associated with intestinal lesions. Beattie, Dickson, Drennan and Glaister all accept that duodenal ulceration does occur, albeit rarely. Martin in his work quotes Curling as having demonstrated ulceration 16 times in 125 cases of burns. On the other hand Gonzales denies having seen them personally, in spite of the many accounts in the literature.

Little is known of the aetiology of the condition. Glaister considers that circulatory changes may be sufficient to cause thrombosis of the venules through slowing of the blood flow, this thrombosis leading to ulceration of the mucosa. Ritchie quotes Harkins' observations on a series of 94 cases of gastric and duodenal ulceration. He enumerates 28 different possible aetiological factors.

At the Zagreb Institute of Forensic Medicine the total number of autopsies performed since 1934 number 10,300. Of these, 240 suffered from burns, males and females being equally represented. Adults totalled 80, children 160.

Inspection of the digestive tract showed ecchymoses and tiny ulcers to be more frequent in the mucosa of the stomach than in that of the duodenum. None was found in the small intestine.

The doudenal ulceration was of the greatest interest. Of the 240 cases of burns, 34 (14 %) showed some ecchymoses in the mucosa of this organ, and 12 (5% of the total) showed between 1 and 3 ulcers, each measuring 2 cm. across. The remaining 194 cases showed no change. There were 8 children (under the age of 9) and 4 adults.

All cases showing duodenal ulceration lived from 4 to 14 days following the burn. No such ulceration was found in those who succumbed in the first 3 or 4 days. A certain minimal time seems to be necessary for the tissue changes to take place if ulceration is to occur.

One case is described in detail. It was that of a 3-year-old boy with third degree burns covering about 50% of the total skin surface. They had been caused by boiling water. He was admitted to hospital immediately, and given treatment for the burns. There was never any loss of consciousness. On the fourth day the temperature rose to 100° F., and on the fifth day he was desperately ill, with gross melaena. He died on the sixth day, and at autopsy typical third degree burns of the skin were found.

In passing it should be noted that an adult can withstand relatively more extensive burns of the skin than can a child, and that it is the extent rather than the depth of the lesions which determines the final outcome.

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Internal examination disclosed a stomach which was distended with clotted blood (Fig. 1); and both the large and the small intestine were filled with blood which was partially clotted, partially fluid.

Further dissection revealed 2 recent ulcers in the first part of the duodenum, 1 cm. from the pylorus. One, about 2 x 3 cm., lay on the posterior wall where it had penetrated the pancreas; in its floor lay exposed a large muscular artery. The other was smaller, measuring 2 x 1 cm., and lay on the anterior wall, in apposition with the gall bladder, which was loosely adherent to the organ. A marked anaemia was evident throughout the body, and the cause of death was given as *effusio sanguinis interna acuta.*

This case is described in some detail as it is the only one of the 240 cases of burning in our records where death has been directly attributable to haemorrhage caused by a perforated duodenal ulcer. It is the more significant because the boy was under hospital supervision the whole time,

and received immediate first aid and general treatment. Nevertheless, duodenal ulceration occurred. It is also notable for the large size of the ulcers, for a diameter of 2 cm. is a maximum in the majority of other cases.

The figures purport to show that such duodenal ulceration occurring as a complication of burns may have occurred less frequently in the post-war years than in the years before and during the war. The numbers are, however, too small to justify conclusive deductions.

Any apparent improvement since the war is attributed to improved treatment both in hospital and in first-aid.

The apparently higher incidence of ulceration in infants is attributed to their relatively larger surface areas of skin compared with their body weight, and to an accelerated tissue reaction which may be bound up with the higher metabolic rate of children.

Many authors (Jajesuria, Marsden, Deters, Ramsey, Mosquera, Manfrini, Gerudini, Harkins, Keeley, Wigham, Weigel, Kapp, Hartman, Lozano, Hansen and Leitner) have described single cases of gastro-intestinal ulceration occurring as a complication of burns. The minority occurred in the duodenum, and few of these progressed to actual perforation.

From this literature, and from our own experience at the Institute, it is concluded that such ulceration is extremely rare. For this reason the figures from the Institute are presented, together with a description of one case dying from haemorrhage due to the perforation of such an ulcer.

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Applications of Polarography to Analytical Toxicology

III. The Polarographic Determination of Bismuth in the Urine

Charles D. Proctor, Ph.D., F.A.I.C.,* and Y. T. Oester, M.D., Ph.D.†

Department of Pharmacology and Experimental Therapeutics, Graduate School and the Stritch School of Medicine, Loyola University Chicago, Illinois, U.S.A.

The methods which exist at the present time for the determination of bismuth in biological material have either a gravimetric or colorimetric basis for their quantitative determination.[1-6] The theoretical and possible practical advantages of the polarographic method of determination have been pointed out in review and reference works on polarography.[9, 14] With these advantages in mind the experiments to be presented were undertaken. It was realized that the choice of a supporting electrolyte for the polarographic cell solution would be limited by the various methods open for destruction of organic material in the samples to be analyzed. The general method used for lead in a previous report[15] was not applicable because bismuth is not quantitatively precipitated by the precipitating agent used in that procedure. In addition, muffle furnace ashing at convenient temperatures causes loss of the metal. While a variety of procedures have been suggested by a number of authors for this purpose[7, 8] the use of a sulfuric acid–nitric acid wet ashing system was decided upon because of its relative rapidity, its broad general use by other workers, and because the ultimate supporting electrolyte system to which it could be finally brought had been studied carefully by Lingane.[10] It was recognized that the suitability of this procedure would be subject to several contingencies which can be listed below:

1. The possibility of interference by metallic constituents other than bismuth.
2. The possibility of interference by contaminants other than metals.
3. Limiting current variation due to supporting electrolyte system variation.
4. Possible bismuth loss during the process of organic matter destruction.

The general process of wet ashing which was decided upon involves treatment of the sample with sulfuric acid and nitric acid followed by careful digestion to the point where sulfur trioxide fumes are liberated, cooling and

* Associate Professor of Pharmacology and Experimental Therapeutics.

† Professor of Pharmacology and Experimental Therapeutics and Acting Chairman of the Department.

subsequent addition of nitric acid. Re-digestion is repeated in the same manner until a colorless solution indicates destruction of the organic matter. The traces of nitric acid are then removed by digesting the residue with water. In the plan evolved the sulfuric acid residue would then be washed from the flask into a suitable volumetric container, and after making the system up to a given volume the process of polarography would be undertaken following the usual nitrogen deareation.

The possibility of other metals interfering was eliminated by reference to the literature,[10] and experimental check of the work presented there. The second factor of interference, that of other contaminants, might occur from two sources, viz. the incomplete destruction of organic matter and the traces of nitric acid which might not have been removed from the sulfuric acid residue. The first of the two possibilities is a function of the ability of the operator and was not found to give interference when the digestion was actually complete. In order to ascertain the effect of the latter possibility it was decided to study the effects of various quantities of nitric acid on the basic polarographic cell solution. In view of the fact that sulfuric acid concentration is a possible variant in the procedure, it was decided that this should be studied in a like manner.

The rationale for such an approach stems from fundamental polarographic theory and fact and is demonstrated by the Ilkovic-Heyrovsky equation. It was first shown by Ilkovic[11] and later confirmed by Lingane and Kolthoff[12] that the diffusion current at the dropping mercury electrode is expressed completely and quantitatively by the following equation:

$$i_d = KnD^{\frac{1}{2}} cm^{\frac{1}{3}} t^{\frac{1}{6}}$$

i_d is the diffusion current, n is the number of unit molar electron equivalents involved, D is the diffusion coefficient, c is the concentration of the reactant involved, m is the rate of mercury flow, t is the drop time, and K is a constant. With the values for m and t being experimentally determinable and c controllable, it becomes apparent that a calculated value for i_d can be obtained if the value of $KnD^{\frac{1}{2}}$ is available. Lingane,[10] using the fundamental differential equation for diffusion to the dropping mercury electrode, calculated the diffusion coefficient of bismuth and proved that the usual value of 605 for the constant K held for bismuth determinations in sulfuric acid over a temperature range of 15-40° C. Lingane's value of 4·31 for the quantity $KnD^{\frac{1}{2}}$ in bismuth–sulfuric acid systems was reproduced by the authors within $\pm 0{\cdot}03$ using the form of the equation shown below:

$$KnD^{\frac{1}{2}} = \frac{i_d}{cm^{\frac{1}{3}} t^{\frac{1}{6}}}$$

Simple rearrangement of the equation allows for concentration check after the values for $m^{\frac{1}{3}}$ and $t^{\frac{1}{6}}$ have been obtained by experiments.

$$C = \frac{i_d}{KnD^{\frac{1}{2}} m^{\frac{2}{3}} t^{\frac{1}{6}}}$$

All changes necessary for the determination of the adherence of the bismuth limiting current to the determinable factors of the Ilkovic equation, under the conditions of slight variability of supporting electrolyte concentration which might result from the method as outlined, were carried out. In order to check for bismuth loss during the wet ashing process a 're-polarographing' procedure was performed. This involved polarographing a set of bismuth solutions of various concentrations made up using the basic electrolyte system designed for the over-all determination. These samples were then processed by wet ashing as previously described, wet ashing in a bismuth free urine, and repeating the polarograph determination after appropriate adjustment to the original dilution.

Materials and Methods

Experimental data, consisting of bismuth determinations in the presence of supporting electrolyte variations, were obtained according to the plan of Kolthoff and Orlemann.[13] These values were used to test the bismuth determinations in relation to exact quantitative methodology. All determinations were made at a controlled temperature of 27° C. and at a constant applied electromotive force of −0·05 V vs. the S.C.E. This is the half-wave potential of bismuth in both sulfuric and nitric acid supporting electrolytes. The drop rate of the mercury in the sulfuric acid supporting electrolyte was determined by averaging the results of 10 determinations made by counting the drops formed in a one-minute interval of time with a manual tabulator, the time interval being controlled by use of an alarm interval timer. The rate of flow of the mercury was ascertained gravimetrically under the same conditions using pre-weighed electrolysis cells which were re-weighed at the conclusion of the one-minute time interval. Calculation of the values of the Ilkovic experimental constants $m^{\frac{2}{3}}$ and $t^{\frac{1}{6}}$ from the average values obtained in the experiments so performed gave 1·64 mg. per sec. and 1·33 seconds respectively.

On the same day that the values of the Ilkovic m and t were determined, using the same capillary dropping electrode, and electrolysis solutions of equal volume to those used during determinations of the constants at a temperature of 27° C., the solutions indicated in Table I were polarographed at voltage settings designed to include the bismuth polarographic wave. All the solutions used were comparable so that they could be referred and compared to the basic supporting electrolyte system consisting of 6 μ gm. of bismuth per c.c., 3 c.c. of concentrated sulfuric acid, 1 c.c. of 1 : 500 gelatin solution, and re-distilled water to make 20 c.c. The variations in nitric and sulfuric acid content were set so as to approximate the possible supporting electrolyte system variation which might be encountered at the end of the wet ashing process proposed for the destruction of organic matter in the analytical method. The results of the experiments as shown in Table I indicate a maximum error of −0·37 μ gm. in the concentration of the solution as determined by the Ilkovic equation and a general average error of $\pm$0·25 μ gm. under the system variations studied. These results seemed to indicate that the possible supporting electrolyte system variation which would be present, in the solutions made up for polarography following the wet ashing process, would be factors of minor importance in creating error in the use of the determination for toxicological purposes. With the matter of the supporting electrolyte system established, the experimental method and procedure for the wet ashing process was outlined and tested.

Table I: Effect of supporting electrolyte variation upon Ilkovic constants in the bismuth–sulfuric acid system

Variation in Basic System	Step Height (mm.)	d (μ amp.)	Bi Ilkovic (mM.)	Bi Present (mM.)	Bi Ilkovic (μ gm. per c.c.)	Bi Present (μ gm. per c.c.)	Difference Bi Present and Ilkovic Bi	Electrode Data
1. + 0·1 c.c. HNO_3								
(a)	64·0	·256	·0273	·0287	5·71	6·00	−0·29	
(b)	67·0	·268	·0287	·0287	6·00	6·00	0·00	
(c)	66·0	·264	·0281	·0287	5·88	6·00	− 0·12	
								$\frac{1}{2}$ KnD is 4·31
								$\frac{3}{4}$ m is 1·64
								$\frac{1}{4}$ is 1·33
2. + 0·2 c.c. HNO_3								
(a)	65·0	·260	·0276	·0287	5·76	6·00	− 0·24	
(b)	68·0	·272	·0290	·0287	6·06	6·00	+ 0·06	
3. 2 c.c. H_2SO_4 instead of 3 c.c.								
(a)	63·0	·252	·0269	·0287	5·63	6·00	− 0·37	
(b)	65·5	·262	·0279	·0287	5·86	6·00	− 0·14	
4. 1 c.c. H_2SO_4 instead of 3 c.c.								
(a)	68·0	·272	·0290	·0287	6·06	6·00	+ 0·06	
(b)	68·0	·272	·0290	·0287	6·06	6·00	+ 0·06	
5. + 0·2 c.c. HNO_3, H_2SO_4 1 c.c. only								
(a)	69·0	·276	·0294	·0287	6·15	6·00	+ 0·15	
(b)	66·0	·264	·0281	·0287	5·88	6·00	− 0·12	
6. Basic system, no change								
(a)	67·0	·268	·0285	·0287	5·95	6·00	− 0·05	
(b)	67·5	·270	·0287	·0287	6·00	6·00	0·00	

Experimental

Reagents:

1. Nitric Acid, A.C.S., Sp. Gr., 1·42.
2. Sulfuric Acid, A.C.S., Sp. Gr., 1·84.
3. Metal-free water. Laboratory distilled water is re-distilled from an all-Pyrex system twice.
4. Gelatin solution: 1·0 g. of gelatin, C.P., is placed in 500 c.c. of metal-free water and the whole well shaken.

Procedure for Urine Determinations

Fifty to 100 c.c. of urine are placed in an 800 c.c. Kjeldahl digestion flask. Three c.c. of concentrated sulfuric acid and 10 c.c. of concentrated nitric acid are added. The solution is digested under the hood until fumes of sulfur trioxide are evolved. (Sulfur trioxide evolution indicates complete use of the nitric acid in the oxidation process and subsequent decomposition of sulphuric acid.) This should not be permitted to occur for any great length of time because it causes supporting electrolyte variation in the final polarograph solution and also because it may cause some bismuth loss. The solution is allowed to cool whereupon 5 c.c. of nitric acid are added and the digestion resumed. The digestion is carried to the point of sulfur trioxide evolution, allowed to cool, and the process of nitric acid addition with subsequent digestion is repeated until the organic matter has been

destroyed as evidenced by a nearly colorless (very pale yellow) or colorless solution. Following the final nitric acid–sulfuric acid digestion the material is allowed to cool and 20 c.c. of metal-free water are used to wash down the neck of the flask. The material is digested to the point of fuming, cooled to room temperature, and the washing and subsequent digestion processes are repeated. This process removes any excessive amounts of nitric acid remaining from the oxidative digestive procedure. After the material has cooled to room temperature following the second digestion with re-distilled water, 5 c.c. of re-distilled water are added to the residue and the solution is poured into a 20 c.c. Pyrex volumetric flask, via a Pyrex funnel. The digestion flask is washed out with two successive portions of 5 c.c. of metal-free water, the washings being added to the volumetric flask. The flask is allowed to cool to room temperature and 1 c.c. of 1 : 500 gelatin solution is added. The solution is then made up to the 20 c.c. volume mark, nitrogen passed through for 5 minutes, and polarographed at settings to include the wave of bismuth in sulfuric acid supporting electrolyte. (Half-wave potential at $-0{\cdot}05$ V vs. S.C.E. $\pm 0{\cdot}02$ V.)

Three solutions, each of a total volume of 40 c.c., containing 6 c.c. of concentrated sulfuric acid, 2 c.c. of 1 : 500 gelatin, but varying in their bismuth concentration so that they contained 5, 10 and 15 μ gm. per c.c. respectively, were made up and divided into two equal 20 c.c. portions. The division was effected with calibrated volumetric pipettes. One of the two portions of each solution was polarographed in the usual manner. The other portion was added to 100 c.c. of normal human urine which had been shown to be bismuth-free by a blank determination made on it. The analytical procedure outlined above was carried out on the solutions thus obtained in full, except that sulfuric acid addition was not necessary because of its prior inclusion in the solution. This procedure yielded two polarograms on each of the original solutions, one taken on the solution in its original form and the other obtained after the solution had gone through the analytical procedure for bismuth in the urine. (Figure 1)

Fig. 1. Polarograms of bismuth before (A) and after (B) being carried through the procedure for the analysis of the metal in urine.

Results typical of three such experiments are shown in Table II and the findings indicate that the process causes loss of trace amounts of bismuth of the magnitude of one-tenth to one-hundredth microgram quantities. Comparison with other methods used to toxicological assay shows that this loss of material being determined is certainly not large enough to negate the usefulness of the method in toxicological analysis.[7, 8]

Table II: Step heights of bismuth polarograms before and after application of the method for bismuth in the urine

Original Solution Concentration (μ gm. per c.c.)	*Step Height Original Solution (mm.)*	*Step Height Test Solution (mm.)*	*Difference (mm.)*	*Difference in Concentration (μ gm. per c.c.)*
5	33	30	−3·0	·45
10	67	65·5	−2·5	·23
15	100	99	−1·0	·15

Apparently the incidence of bismuth intoxication has been steadily on the decline. This statement is made as a result of the decrease in the reported cases and requests for toxicological bismuth diagnosis in the toxicology laboratory of the Cook County Hospital, Chicago, Illinois. As a result of this circumstance, the clinical material available for study of the application of the polarographic analysis of bismuth in urine was limited to a study of 3 cases. All of these were intoxications from over-zealous bismuth medication. Consequently the study of the application of the method consisted of duplicate determinations of the bismuth concentrations present in the diagnostic 24-hour urine samples taken from the patients. The data entered in Table III indicate that satisfactory results are obtained in duplicate runs of the same 24-hour urine containing bismuth.

Table III: Duplicate Polarographic Determinations for Bismuth made on Urine Samples from Patients with Bismuth Intoxication

Case	*Volume Urine Used per Sample Tested*	*Bismuth Found (μ gm. %)*		*Difference (μ gm. %)*
		Sample 1	*Sample 2*	
1	50 c.c.	280	272	8
2	100 c.c.	31	29	2
3	100 c.c.	58	58	0

Discussion

Previous methods for the determination of bismuth in biological material have all been hampered by certain disadvantages. In many cases, the oxidative destruction of the organic matter present in the biological material of inherent necessity to the method, involved the use of perchloric acid. This substance is always an explosion hazard.[4, 7] Other methods have introduced exacting control of pH values at various steps of the operation. Error is likely during their execution in the hands of the inexperienced.[2] Some of the procedures involve time-consuming precipitations, gravimetric weighing and extraction processes.[2, 3, 6, 7] It is apparent, in the process of the use of the polarographic method and during the experimental verification of its quantitative application, that the majority of these disadvantages are not present or troublesome in that procedure.

Summary

1. A method for the determination of bismuth in the urine by the polarographic method of analysis has been presented.

2. Analysis of the method presented has demonstrated that it is quantitative and is not subject to serious error due to the possible variations of the supporting electrolyte which might occur from application of the method.

3. Application of the method has been demonstrated by determinations carried out on 24-hour urine samples from patients with bismuth intoxication.

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Vicarious Liability for Medical Negligence*

M. A. Millner, B.A., LL.B. (Rand), B.C.L. (Oxon.)
University of the Witwatersrand, Johannesburg

It is not the primary purpose of this article to discuss the nature of legal liability for negligent conduct generally nor the personal liability of a doctor in respect of his own negligent behaviour, but rather the liability of a doctor or hospital in respect of the negligence of staff or subordinates, that is to say, vicarious liability.

The ground may be cleared by some general observations on the principles upon which, in law, liability may be incurred in such cases. Liability may be founded on (1) breach of contract or (2) delict (civil wrong).

(1) *Breach of Contract.* The relationship between a hospital or a doctor and a patient is normally a contractual one—the contract being for the rendering of medical services in return for a fee. The terms of the contract could be quite specific so as to place beyond doubt what liabilities the hospital or doctor assumes towards the patient. This in practice is rare. Usually there is no writing and no formal terms are discussed defining the extent of the hospital's or doctor's obligations, which are therefore left to be inferred upon the principles of the common law.

At common law, the parties are bound not only by the express terms of their agreement but also by implied terms. However,

> 'the Court ought not to imply a term merely because it would be a reasonable term to include if the parties had thought about the matter, or because one party, if he had thought about the matter, would not have made the contract unless the term was included; it must be such a necessary term that both parties must have intended that it should be a term of the contract, and have only not expressed it because its necessity was so obvious that it was taken for granted' (Wessels, *Law of Contract*, para. 279).

Thus if the patient relies on an implied agreement to make the hospital or doctor liable to him for the negligence of a subordinate he will need to show that it was a necessary term of the contract (in the sense defined above) that there should be such liability.

(2) *Delict.* Independently of contract, a man is generally answerable in law for injuries which he negligently causes to another. Negligence means failure to exercise reasonable care. If a reasonable man would, in the circumstances, have foreseen the likelihood of harm and guarded against it, then the failure so to guard against that harm is negligence and, if the harm materializes, the injured person is entitled to claim damages to com-

* Based on an address given by the author to the South African Medico-Legal Society, Johannesburg, on 17 June 1957.

pensate him for his injuries. Applied to a medical practitioner, this means that he 'is not expected to bring to bear upon the case entrusted to him the highest possible degree of professional skill, but he is bound to employ reasonable skill and care' (*Mitchell* v. *Dixon,* 1914 A.D. 519 at 525). 'And in deciding what is reasonable, the Court will have regard to the general level of skill and diligence possessed and exercised at the time by the members of the branch of the profession to which the practitioner belongs' (*van Wyk* v. *Lewis,* 1924 A.D. 438 at 444). Thus the law sets reasonable and not impossibly high standards of professional skill and care; as Denning, L.J., observed in *Roe* v. *Minister of Health* [1954] 2 Q.B. 66: 'We should be doing a disservice to the community at large if we were to impose liability on hospitals and doctors for everything that happens to go wrong. . . . We must insist on due care for the patient at every point, but we must not condemn as negligence that which is only a misadventure' (p. 926).

Ordinarily a man is only answerable for his own negligence in this way. However, probably for reasons of social policy, the law recognizes the *vicarious* liability of an employer for the negligence of his employees even though the employer is himself quite free from negligence and exercised proper care in the selection of his employees for the task in hand. It is customary to say that this doctrine of vicarious liability applies only where there exists between employer and employed the relationship of master and servant. Consequently if a master-servant relationship exists between the hospital on the one hand and a doctor or nurse on the other, the hospital might be made liable for the wrongdoing of that doctor or nurse on this principle. It will be seen, however, that the question whether there is such a relationship of master and servant between the hospital and its subordinates, and if so to what extent, raises controversial problems.

Liability of Hospitals

It is clear that a hospital contracts with its patients to use proper care in the selection of a competent staff and to place at the disposal of that staff fit and proper apparatus and appliances. If a patient were injured in consequence of the hospital's failure in any of these respects, he would plainly have a good cause of action—the hospital would itself be liable in respect of its own negligent conduct. But what if a fully competent member of staff acts negligently towards a patient in a particular matter thereby injuring him? Is it the hospital's obligation to nurse the patient skilfully—in which case it is liable—or is it merely to take on a skilful nurse—in which case it is not?

Ordinarily if X contracts to do something for Y and chooses to do it through his servants, he is answerable to Y for the negligence of his

servants; that is implied in the contract. Similarly X is answerable to Y in delict for the negligence of X's servants. Nevertheless in *Hillyer* v. *Governor of Saint Bartholomew's Hospital,* 1909 2 K.B. 820, the English Court of Appeal refused to make a hospital liable to a patient who was negligently injured whilst under an anaesthetic in the operating room. The patient was being examined by the consulting surgeon attached to the hospital, assisted by an anaesthetist, nurses and carriers. During the examination the patient's left arm came in contact with a hot water tin beneath the table and was injured thereby, and his right arm was bruised by pressure. Referring to the nurses and carriers, Farwell, L.J. said:

'If and so long as they are bound to obey the orders of the defendants, it may well be that they are their servants, but as soon as the door of the theatre or operating room has closed on them for the purpose of an operation (in which term I include examination by the surgeon) they cease to be under the orders of the defendants and are at the disposal and at the sole orders of the operating surgeon until the whole operation has been completely finished; the surgeon is for the time being supreme and the defendants cannot interfere with or gainsay his orders'.

This reasoning is, as we shall see presently, open to criticism. But at least it puts the decision on a fairly narrow basis. Kennedy, L.J., however, approached the matter differently and drew a distinction which had far-reaching consequences. He distinguished between matters of professional skill and 'purely ministerial or administrative duty, such as, for example, attendances of nurses in the wards, the summoning of medical aid in cases of emergency, the supply of proper food, and the like'. Whilst a hospital was answerable for the due performance by its staff of their ministerial or administrative duties it was not so answerable 'in matters of professional skill in which the Governors of the hospital neither do nor could properly interfere either by rule or by supervision'.

This means, in short, that in regard to the professional duties of its staff, a hospital escapes the liability which normally attaches to a contracting party who chooses to use servants for the performance of his contract. This special immunity appears to rest on one of two grounds: either (1) the hospital staff, while acting professionally, are not servants at all—an argument which will receive attention presently—or (2) even though they are servants, there is a special implied term in the contract between hospital and patient exempting the hospital from liability for the negligence of its staff when engaged in professional duties.

This latter ground for exonerating the hospital can hardly be sustained. Professor A. L. Goodhart long ago pointed out (See Vol. 54 (1938) *Law Quarterly Review* at p. 560) that the fact that a servant is required to exercise professional skill does not, as a general rule, give rise to any such special implied term. He instances the aviation company which employs an aviator to fly a plane on a difficult course or the steamship company

which employs a certificated shipmaster to navigate its ship; the employers are liable in such cases even though they cannot tell their servants how to perform the tasks they have employed them to do and would in fact make themselves criminally liable if they attempted to do so. 'Why then,' asks Goodhart, 'should a special term be implied in the case of a trained nurse who exercise less professional skill than does an aviator or shipmaster?' There being no rational answer to this question, the supposed immunity of the hospital would appear to depend upon the validity of the first of the two grounds mentioned above, namely the contention that the staff, when acting professionally, are not servants at all.

Hillyer's case and Kennedy, L.J.'s judgment were followed in a number of cases both in England and South Africa. *Lower Umfolosi Hospital* v. *Lowe,* 1937 N.L.R. 31, will serve as an example. In that case the patient, whilst recovering from an anaesthetic after an operation in the Lower Umfolosi Hospital, was severely burned by a hot water bottle owing to the negligence of a nurse. The Court held, applying the distinction made by Kennedy, L.J., in *Hillyer's* case, that the placing of the hot water bottle in the patient's bed and the subsequent supervision of him were professional duties on the part of the nurse and that the hospital was therefore not liable. Decisions such as this left *Hillyer's* case in unchallenged possession of the field and it seemed to be established law in South Africa that a hospital was exempt from liability for the negligence of its doctors or nursing staff so long as that negligence was in the course of 'professional' as opposed to 'administrative' duties; for—so it was reasoned—only in the latter case did a master-servant relationship exist.

Now it is not at all clear where administrative duties stop and professional duties begin; but virtually all a doctor's duties must surely be regarded as professional and, to judge by the application of the distinction in *Lowe's* case, most of a nurse's duties would be so too. Thus on this basis the injured patient's prospects of success in a law suit against the hospital would be slender indeed.

Fortunately there are good reasons for thinking that our courts, if faced with this issue afresh, would discard this approach.* As we have seen, the justification for it is the view that the hospital staff, whenever engaged on professional duties, do not rank as servants of the hospital which employs them; that is, there is no master-servant relationship between them, but the servants are rather like independent contractors for whose acts the hospital bears no responsibility. It is believed that this view does not bear examination.

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* Perhaps it is a portent that in the recent case of *Esterhuizen* v. *Administrator, Transvaal* (1957 (3) S.A. 710) in which a hospital was held liable for the wrongful act of its radiologist, the '*Hillyer* defence' was not raised.

The Master-Servant Relationship

What is the legal test of the master-servant relationship? In the traditional view, a servant is one who is subject to the orders and control of his employer not only as to the work which he is to do but also as to the manner of doing it, as contrasted with an independent contractor, who uses his own discretion as to the manner of performance of the specified work. On the argument that the mode of performance of professional duties is not susceptible of control, the conclusion was reached that hospital staff, whilst engaged in professional duties, could not be servants.

But this argument has lost favour with the English courts, which have in recent years abandoned the standpoint of *Hillyer's* case, or at any rate the reasoning of Kennedy, L.J. Thus in *Gold* v. *Essex County Council* [1942] 2 K.B. 293, a radiographer negligently failed to cover a patient's face with a lead-lined rubber cloth, using instead only a piece of lint, with the result that the patient's face was permanently disfigured. The hospital was held liable. Lord Greene, M.R., after preferring the judgment of Farwell, L.J., in *Hillyer's* case, said of it (at p. 299): 'I can find nothing in his judgment to support the view that the authorities of a hospital are not liable for the negligent act of a nurse in the performance of her general duties as a nurse, *be they duties requiring professional skill or duties of a purely administrative or domestic nature such as giving a patient his meals* (writer's italics) . . . I should myself have thought that the true ground on which the hospital escapes liability for the act of a nurse who, whether in the operating theatre or elsewhere, is acting under the instructions of the surgeon or doctor is, not that *pro hac vice* she ceases to be the servant of the hospital, but that she is not guilty of negligence if she carries out the orders of the surgeon or doctor, however negligent those orders may be'. Further on in his judgment (see p. 301) Lord Greene states: 'It appears, therefore, that the supposed limitation on the liability of hospitals in respect of members of their staff (other than doctors and surgeons) when not acting under the direct instructions of doctors or surgeons owes its origin entirely to the views expressed by Kennedy, L.J., in *Hillyer's* case with such support as is to be gathered from the concurrence of Lord Cozens-Hardy, M.R. There is, so far as I am aware, no English authority which prevents this court from giving effect to its own opinion on the question which arises in the present case . . . the first task is to discover the extent of the obligation assumed by the person whom it is sought to make liable. Once this is discovered, it follows of necessity that the person accused of a breach of the obligation cannot escape liability because he has employed another person, whether a servant or agent, to discharge it on his behalf, and this is equally true whether or not the obligation involves the use of skill'.

Collins v. *Hertfordshire C.C.*, 1947 K.B. 598, carries still further the trend in *Gold's* case. In *Collins'* case, the operating surgeon gave telephonic directions to the resident house surgeon (a final year student) to administer 1% procaine with 1 : 200000 adrenalin. The resident surgeon misheard the directions and prepared '1% cocaine', which the surgeon injected, believing that he was injecting a perfectly proper and harmless local anaesthetic. The injection being lethal, the patient died. The Court held that the hospital was vicariously responsible for the negligence of the resident surgeon; but not for that of the visiting surgeon since in the case of the resident junior house surgeon they had, and in the case of the visiting surgeon they had not, the power to direct him or her what to do and how to do it.

The next stage is marked by *Cassidy* v. *Ministry of Health* [1951] 2 K.B. 343. The patient there underwent an operation on his left hand which necessitated post-operational treatment, during which he was under the care of (*a*) the surgeon who operated, who was a whole-time assistant medical officer of the hospital, (*b*) the house surgeon and (*c*) members of the nursing staff. All of these persons were under contracts of service with the hospital. When the hand was released from the splint it was found that it was to all intents and purposes useless and after two unsuccessful manipulative operations, all attempts to remedy the condition were abandoned. The injury was found to be due to negligence in the post-operational phase. It was not clear whether the negligence had to be imputed to the whole-time assistant medical officer, the house surgeon or one of the nurses, but the three members of the Court agreed that it was unnecessary to investigate which one was to blame, as the hospital was liable in any case. Thus this case affirms the liability of a hospital for the negligence of all members of its permanent medical staff.

The latest stage reached in England is illustrated by *Roe* v. *Ministry of Health* [1954] 2 Q.B. 66, in which nupercaine, a spinal anaesthetic, was injected into two patients by a specialist anaesthetist assisted by the theatre staff. The anaesthetist carried on a private practice but was under an obligation to provide a regular service to the hospital, for which he received certain remuneration. The nupercaine had been contained in sealed glass ampoules stored in a solution of phenol. In fact, the phenol had percolated into the ampoules through invisible flaws in the glass, causing both patients to develop severe symptoms of spastic paraplegia, resulting in permanent paralysis from the waist down. It was held that in view of the prevailing medical knowledge in 1947 such a danger would not be anticipated by 'the ordinary anaesthetist in this country' and therefore there was no negligence, but that had negligence been found, the hospital would have been liable for the acts of its part-time specialist in anaesthetics even though he was not a member of the permanent staff.

Thus in England the liability of a hospital has now been recognized in respect of negligence in the professional conduct of its staff not only in the cases of nurses and radiographers but also in the case of resident medical officers, members of its permanent medical staff and part-time specialists in anaesthetics. In *Roe's* case two judges left open the question whether the hospital is answerable for the conduct of visiting surgeons and physicians. Denning, L.J., considered it was, and there is little doubt that this is so.

It has been suggested (by Kahn-Freund (Vol. 14 (1951) *Modern Law Review,* 504)) that the importance of these developments in England is that they are 'based upon a transformation of the traditional "control" test i.e. of the test which is being used in order to distinguish between a servant and an independent contractor, and between a contract of employment (or of service) and a contract for work and labour (or "for services").' He suggests that this distinction is based upon the social conditions of an earlier age which assumed that the employer was able to direct and instruct the labourer as to the technical methods he should use in performing the work. 'The control test was well suited to govern relationships like those between a farmer and an agricultural labourer (prior to agricultural mechanization), a craftsman and a journeyman, a householder and a domestic servant, and even a factory owner and an unskilled "hand". . . . The technical and economic developments of all industrial societies have nullified these assumptions. The rule *respondeat superior* . . . "applies even though the work which the servant is employed to do is of a skilful or technical character, as to the method of performing which the employer himself is ignorant"—per MacKinnon, L.J., in *Gold's* case, at p. 244. To say of the captain of a ship, the pilot of an aeroplane, the driver of a railway engine, of a motor vehicle or of a crane that the employer "controls" the performance of his work is unrealistic and almost grotesque.' In Kahn-Freund's view 'the development is in the direction of something like an "organization" in preference to a "control" test. In future the question may perhaps be: "did the alleged servant form part of the alleged master's organization?" rather than "was he subject to his orders in the actual doing of his work"?' Other writers, such as Grunfeld (Vol. 17 (1954) *Modern Law Review,* 550) also speak of 'the bankruptcy' of the control test in contemporary conditions of employment and of the 'important attempts to re-establish the legal relationship of master and servant upon an acceptable modern footing'.

Can South African courts follow this line of development or is the 'control' test of the master-servant relationship too deeply entrenched in the law to be altered otherwise than by legislative change? There is no doubt that it is the test habitually adopted by the courts (see *Colonial Mutual Life Assurance Society* v. *Macdonald,* 1951 A.D. 412; *Dukes* v. *Marthinu-*

sen, 1937 A.D. 12) but perhaps it is possible to argue that the control test is only one criterion—albeit a very important one—of the existence of a master-servant relationship or of a contract of service and that it has to be supplemented in a suitable case by the 'organization' or 'subordination' test. At least in English law authority for such a point of view could be found (see Grunfeld's article *supra*, p. 551).

If it is not possible to depart from the control test in our law, on what basis could the liability of a hospital rest? McKerron (*The Law of Delict*, p. 125, note 90) suggests that unnecessarily heavy weather is made of the 'control' test problem. He argues that although a hospital authority could not *directly* interfere with members of the staff in professional matters 'there would seem to be no reason why it could not do so indirectly, if it chose; for example through a senior medical officer appointed by it to supervise the work of the hospital on the professional side'. He therefore submits that 'there is no real difficulty in holding that a hospital authority has the right (in theory at any rate and that is sufficient for the purpose of the test) to control the members of the hospital staff even in matters of professional care and skill'. Nevertheless this solution is open to the criticism of being somewhat artificial, depending as it does upon a fictional control.

An alternative line of argument is to be found in the approach of Denning, L.J., in *Cassidy's* case and *Roe's* case. His Lordship considered that hospitals are under a 'personal' duty to use reasonable care in treating their patients and that although the performance of this duty had to be delegated to employees, the hospital could not thereby rid itself of its obligation and remained responsible for the negligence of its employees whether they were servants in the strict sense or not: 'I think that the hospital authorities are responsible for the whole of their staff, not only for the nurses and doctors but also for the anaesthetists and the surgeons. It does not matter whether they are permanent or temporary, resident or visiting, whole time or part time. The hospital authorities are responsible for all of them. The reason is because, even if they are not servants, they are the agents of the hospital to give the treatment. The only exception is the case of consultants or anaesthetists selected and employed by the patient himself' (*Roe's* case, at p. 137).

It is not clear, however, that this approach is consistent with our law, at least as regards delictual obligation. McKerron has therefore suggested (Vol. 74 (1955) *S.A. Law Journal*, 97) that our courts, whilst reaching the same conclusion as Denning, L.J., would base it upon 'a contractual obligation because 'it is difficult to see how, apart from contract, there can be any question of a hospital being liable for the negligence of a person who is merely an agent and not the servant of a hospital'. In other words, a term

of this nature would be implied in the contract between hospital and patient as being a 'necessary term' in the sense discussed above. And indeed this contractual approach would seem to offer a reasonable solution of the problem. But even a delictual solution, although rather forced, is not entirely out of the question. McKerron's rejection of it possibly overlooks the fact that our law *has* recognized certain exceptional cases where an employer of an independent contractor does incur liability in respect of harm caused by the latter's negligence. One such exceptional case is where the work which the contractor is employed to do is '*per se* dangerous'. In such a case the employer 'has not merely a duty to take care but a duty to provide that care is taken'—per Langton, J., in the *Pass of Ballater,* 1942 P. 112, at 117. (See *Dukes* v. *Marthinusen,* 1937 A.D. 12). In substance, the rule for which Denning, L.J., is contending appears to coincide with this. In other words it might be argued that the work which the hospital carries out through its agents is '*per se* dangerous' in as much as the health and life of the patients are at stake and that consequently it cannot, by means of delegation, relieve itself of responsibility for the manner in which its agents carry out that work. One must however concede that the '*per se* dangerous' doctrine is at the best of times not quite at home in our law. It is instanced typically by dangerous works near a highway and its translation to the hospital ward is a novelty likely to provoke some judicial resistance.

Doctor's Liability to Patient

A doctor is of course personally liable in respect of his own negligence jointly and severally with the hospital which employs him. He would be liable also for acts done by subordinates, including nurses, in pursurance of his own negligent instructions. And the subordinate, for example the nurse acting on the doctor's instructions, could scarecly be said to be negligent herself unless of course the instructions were patently negligent.

What is the position when a nurse, whilst under the general control of a doctor, as is the theatre sister during an operation, carries out her duties negligently? In *van Wyk* v. *Lewis,* 1924 A.D. 438, the defendant was a surgeon who had performed an urgent and difficult abdominal operation upon the plaintiff, assisted by an anaesthetist and a qualified nurse on the hospital staff who acted as theatre sister. One of the swabs was overlooked and remained in the patient's body. The evidence showed that, in accordance with the usual practice at that hospital, the surgeon had relied upon the sister to count and check the swabs used and that at the end of the operation he had made as careful a search as the critical condition of the patient permitted.

The court held that the surgeon had not himself been negligent in the circumstances* and that even assuming that the sister had been negligent, the surgeon was not vicariously liable in respect thereof. Speaking of the 'independent part in the operation played by the sister', Innes, C.J., said: 'she was not the servant of the respondent; she was under his general control during the operation, but she was also a collaborator to whom, as already pointed out, it was reasonable to entrust the work of counting and checking the swabs'. Wessels, J.A., said (at p. 458): 'the relation of a hospital sister or nurse in a public hospital to a surgeon operating in that hospital is not that of master and servant nor is it analogous to such a relationship. The sister or nurse is an independent assistant of the surgeon though under his control in respect of the operation. . . . It is true that during the actual operation it is the duty of the hospital sister or nurse to do what the doctor requires of her in the same way as it is the duty of an assistant surgeon to act under the principal surgeon's instructions but it cannot be contended that such an assistant surgeon is a servant of the operating surgeon. The truth is that hospital sisters and nurses form a distinct branch of the hospital. They are members of an allied profession and have duties of their own to perform. They are subordinate to the surgeons but they are in no way their servants. The surgeon is not responsible for what the nurse does in the sense that a master is responsible for the acts of his servant. The surgeon does not insure that he will be responsible for every misfeasance of the nurse. To make him so would make his position intolerable'. Referring to *Hillyer's* case (in which, it will be remembered, Farwell, J., treated the nurse and sisters as, *pro tempore,* servants of the operating surgeon since they took their orders from him alone), Wessels, J., went on to say: 'They (i.e. the nurses) cease for the time being to be the servants of the corporation: but it is a far cry from that to say that the nurses become the servants or even analogous to servants of the operating surgeons. . . . In many matters the nurse acts on her own initiative and it is her duty to do so. Though she is under the surgeon's direction she is there as an assistant not as a servant or agent.'

The difficulty here is of course the control test of the master-servant relationship. On that test it is arguable that the nurse is *pro tempore* the servant of the doctor and that the doctor is not the servant of the hospital. But *van Wyk's* case shows that the court is not prepared to regard the nurse in such cases as the servant of the doctor, even using the control test. Whilst this view commends itself in regard to the relation of surgeon to nurse, it creates a difficulty of its own by presenting the nurse as a servant without any master at all. This dilemma is, however, at bottom

* It does not follow, of course, that a surgeon who fails to ensure that all swabs are removed will necessarily escape liability; cf. *James* v. *Dunlop,* reported in Brit. Med. J. 1931, Vol. 1, p. 730, in which the surgeon was held to be negligent in such a case. This turns on the full circumstances.

a product of the false distinction between professional and administrative duties and must yield to the view expressed by Denning, L.J., in *Cassidy's* case (p. 261) that the nurses remain the servants of the hospital authorities even where they are under the direction of the surgeon. The 'subordination' test makes the matter much plainer, for it is then apparent that both the doctor and the nurse are part of the organization of the hospital for carrying out the treatment of the patient; they are fellow servants of the hospital and in so far as either of them is negligent the hospital is vicariously answerable.

It should be observed that the arguments for making a hospital liable for the negligence of its staff would apply equally to making a doctor liable for the negligence of his own staff. But a doctor's staff would scarcely include someone called in by him to consult or someone to whom he has referred a patient. Thus in the case of *Lymbery* v. *Jefferies,* 1925 A.D. 236, where a doctor sent his patient to a certain radiographer for X-ray treament, it was held that the doctor was not liable for the radiographer's lack of skill and care in administering the treatment. Wessels, J.A., delivering the judgment of the Court, said: 'If a medical practitioner or surgeon advises his patient to be treated by some third person, either because it is a treatment which he cannot carry out himself or which it is customary to entrust to a third person, then the latter does not as a general rule act as the agent of the medical practitioner or surgeon'.

Doctor's Liability to Indemnify Hospital

It is usually said that in every contract between a master and servant there is an implied term that the servant is reasonably skilled and competent for the task he undertakes. The servant is therefore bound to exercise reasonable skill and care about his master's business and any damages which the master may have to pay to a third person in consequence of the servant's breach of this duty flow directly from the breach so that the master is entitled to recover the amount of such damages from the delinquent servant. In England this standpoint has recently received the approval of the House of Lords (*Romford Ice and Cold Storage Co. Ltd.* v. *Lister,* 1955 3 All E.R. 460 (C.A.)) and it is generally thought that our courts would take the same view. A doctor would therefore be obliged to make good to the hospital the loss which his negligence had caused it.

The majority of the judges in *Lister's* case considered that the fact that the employer carried insurance against third-party liability did not exclude the servant's obligation to indemnify him. On this point, a minority dissented, being of the opinion that in such circumstances there was an implied understanding between master and servant that the master would not seek an indemnity from the servant. The reason and justice in this view may

very well commend it to South African Courts in preference to the view of the majority. But as long as the matter remains undecided, it would be an elementary measure of prudence for the servant to protect himself by express terms in his contract of employment.

* * *

The upshot of this discussion is that the negligent injury of a hospital patient entitles the patient to seek redress either from the doctor (or nurse) who was personally at fault, or from the hospital employing him (or her). If the patient elects to claim from the hospital, the negligent person does not necessarily go scot free: save where there is an implied understanding between them to the contrary (as may very well be the case when liability insurance is contemplated) he may yet have to answer to the hospital for what his negligence has cost it.

Notes and News

First South African Medico-Legal Congress

The South African Medico-Legal Society has arranged for this Congress to be held in Johannesburg, from Thursday night, 31 July to Saturday (midday), 2 August 1958.

The *Patron* is the Chief Justice the Hon. Mr. H. A. Fagan. The *President* is Prof. S. F. Oosthuizen (President of the South African Medical and Dental Council). The *Vice-President* is the Hon. Mr. L. Greenberg.

The opening session at 8.15 p.m. on 31 July, will be held at Medical House, 5 Esselen Street, Hospital Hill, Johannesburg.

The sessions for the next day will be held in the new Lecture Theatre of the South African Blood Transfusion Service, corner of Klein and Esselen Streets, Hillbrow, Johannesburg.

The evening session on 1 August (to be devoted to the screening of medico-legal films) will be held in the Harveian Theatre, Medical School, Hospital Street, Johannesburg.

The following topics are scheduled for discussion:

1. *Artificial Insemination.*
2. *A Symposium on Acute Alcoholism.*
3. *Problems of Blood Transfusion.*
4. *A Symposium on Sudden Death in Infancy.*
5. *Problems of Consent to Medical Procedures.*

6. *Two Recent American Films* (in sound) entitled *The Medical Witness* and *The Doctor Defendant,* will be screened, followed by a discussion on the films, and an *Open Forum* on any topic discussed at the Congress.

These films have been sponsored by the Wm. S. Merrell Company of Cincinnati, in co-operation with the American Medical Association and the American Bar Association. They have been made specially available for this Congress by the Wm. S. Merrell Company.

7. *Medico-Legal Exhibits and a Visit to the South African Blood Transfusion Centre.*

The Congress registration fee for members of the South African Medico-Legal Society is 10s. The registration fee for non-members is £1 10s. (The normal subscription for membership of the South African Medico-Legal Society is £2 2s. This includes a free subscription to the *Journal of Forensic Medicine*).

Practitioners who wish to register for attendance at this Congress must advise the Honorary Organizing Secretary not later than 15 July 1958. This will enable adequate accommodation arrangements to be made for the meetings and ensure sufficient time for the printing of a Congress prospectus which, it is hoped, will include the names and addresses of all participants in the Congress.

All communications must be directed to:

Honorary Organizing Secretary (Dr. H. A. Shapiro), First South African Medico-Legal Congress, P.O. Box 1010, Johannesburg.